The Influence of the Resilience of the Arterial Wall on Blood-Pressure and on the Pulse Curve.

By S. Russell Wells and Leonard Hill, F.R.S.

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This communication is the result of two independent but converging lines of research. It is well known that when a fluid is driven with a rhythmically varying pressure through a sufficient length of a distensile elastic tube, the pressure at the exit loses its rhythm and becomes constant and the flow continuous, whereas if the tube is rigid, the pressure at the outlet varies as that at the inlet (less the change due to friction) and the outflow is intermittent.

Since the arteries are distensile elastic tubes and the blood is rhythmically forced into them by the heart, it follows that the curve of blood-pressure must be altered to a greater or lesser degree by the distensibility and elasticity of the arterial wall.

We use the term resilience in this paper to express the ease with which an elastic tube distends with a rise and recoils with a fall in pressure of the contained fluid; thus, a rubber tube with a wall 0·2 mm. thick is more resilient than one with a wall 0·4 mm. thick, the thinner, more resilient tube yields with the rise and recoils with the fall of pressure more than the "harder," thicker walled, less resilient tube. A glass tube in this sense has no resilience, and the same may be said of rubber pressure tubing.

As the arterial wall contains muscle its resilience will be altered by a more or less contracted state; as the degree of contraction and resilience may vary locally it is to be expected that the curve of blood-pressure may also vary, *e.g.* in the brachial and femoral arteries. Further, as the peripheral resistance in any area may alter the tension of the arterial wall, its resilience may vary without any change in the muscular state of the arterial wall.

Observations made by one of us (L. H.) with W. Holtzman and Martin Flack, and later with R. A. Rowlands,* on cases of aortic regurgitation placed in the horizontal position, have shown that the systolic pressure is much higher in the leg than in the arm, *e.g.* 100–150 mm. of mercury higher, and so characteristic is this difference that it is a diagnostic sign of the condition. Thomas Lewis found that the same held good in the case of a dog in which he had experimentally rendered the aortic valves incompetent one month previous to taking the observations.† His measurements were

* 'Heart,' 1909, vol. 1, p. 73; and 1912, vol. 3, p. 219.

† 'Heart,' 1912, vol. 3, p. 222.

recorded by means of cannulæ placed in the arteries and connected with Hürthle manometers. Hürthle* and others have recorded previously higher readings of pressure in the femoral than in the carotid artery of the dog. Tigerstedt ascribed these to reflection of the primary pulse wave without change of sign and addition of the reflected to the primary wave in the femoral artery.† The difference of the systolic pressures in the arm and leg in aortic cases was ascribed by L. H. and his co-workers to the better conduction of the systolic wave crest in the leg arteries, which were assumed to be in a more contracted and harder state.

This view was confirmed by experiment, for it was found, on placing the legs and buttocks of the patients in a hot bath, the difference between the readings of arm and leg arteries was abolished, and this was ascribed to the expanding and softening of the contracted walls of the latter. Also, in the case of healthy young men placed in the horizontal posture, while it was found that the leg and arm readings of systolic pressure were normally the same, these were rendered temporarily unequal after the subjects had run twice up and down a long flight of stairs (particularly if the arm were placed in hot water beforehand); the heart was thereby made to beat forcibly, while the leg arteries became more contracted, so the crest of the wave was better conducted in them than in the arm arteries. By placing one arm in hot water, it was found possible to render the reading different in the two arms, even in the resting subject, much more so after a short period of violent exercise. If the wrist alone were placed in hot water, the radial gave a lower reading than the brachial, but if the elbow were placed in hot water, readings of brachial and radial were equal, both being lower than in the other and cooler arm; bandaging the hand tightly made no difference to the reading.

The conclusion arrived at was that the inequality was due to an altered condition of the arterial wall and not to diminished peripheral resistance, and these experiments led to the conception that the nature of the arterial wall affects the conduction of the systolic wave, and that the blood-pressure, as ordinarily measured by a sphygmometer, by the method of obliteration of the pulse, depends not only on the pressure wave produced by the heart, but also on the effect on this wave of the arterial wall, a new factor which has not hitherto been taken into account.

A difference of pressure between the arm and leg readings has been noted by several observers in cases where the arteries are thickened and hardened as in old people. This difference has been ascribed to an error in the

* 'Arch. f. d. ges. Physiol.,' vol. 47, p. 32.

† 'Lehrb. d. physiol. des Kreislaufes,' 1893, p. 352.

readings due to the thickened artery resisting compression, just as an empty rubber tube does.

One of us (L. H.) and Martin Flack have found that such differences of pressure are lessened by keeping on the pressure of the armlet, and lowering and raising it so as to take several readings of: (1) the reappearance; (2) the disappearance of the pulse. Our explanation is, that the artery cut off from the blood relaxes and softens, and therefore the crest of the systolic wave is diminished. It has been shown by Bayliss that compression of an artery is followed by vascular dilatation in the area cut off from the blood.

In many of these cases the force of the pulse is irregular; now and again an extra large systolic crest forces its way beneath the armlet, and such large waves are better conducted by the leg arteries, just as happens in the case of aortic regurgitation.

By means of a circulatory schema, in which two lengths of artery are inserted, one to be compressed, the other to be palpated (the latter gave the index, the disappearance of the pulse), it was easy to demonstrate that the systolic pressure is read more accurately when the palpated artery is made tense (produced in this schema by obstructing the outflow by means of a mercury valve) than when it is soft. In the first case the readings of systolic pressure taken in the pump and in the artery are the same, in the second case the reading taken in the artery is lower.

In the living animal with its vasomotor nerves, and pressure changes of rapid rate, and output of the heart varying from second to second, it is extremely difficult to study exactly the effect of the various factors on the character of the pulse curve, for one cannot vary at will one of these factors without affecting the others. From these considerations it appeared desirable to one of us (S. R. W.) to investigate the subject by means of non-living elastic tubes. Halls Dally and K. Eckenstein have assisted in this research, which will be published in full later.

After considerable experiment an apparatus was devised, by means of which fluid at a known rhythmically changing pressure could be passed (*a*) through elastic tubes of the same calibre, but with walls of various known thicknesses; (*b*) through various lengths of the same tube, and (*c*) keeping to the same tube, the absolute pressure could be varied, while maintaining the same difference between the systolic maximum and the diastolic minimum, or this difference could also be varied at will.

The tubes used in the experiments were various lengths of rubber tube all of the same internal calibre, but with walls of 0·8, 0·6, 0·4, and 0·2 mm. thickness. The pressure variations of the fluid before flowing through the resilient tube and at the end of it were recorded by Hürthle's manometer.

It was found when the same resilient tube was used, but the diastolic pressure of the entering fluid varied, keeping the interval between the systolic and diastolic pressures as far as possible the same, that the higher the pressure and consequently the more the resilience of the tube was brought into action by stretching, the nearer together were the diastolic and systolic pressures at the end of the resilient tube. In other words the smaller was the amplitude of the pressure waves, and the more closely did the pressure approach to a continuous one. As an instance, the following experimental results may be cited, working with 30 cm. of a rubber tube, the walls of which were 0·8 mm. thick and recording the pressures in millimetres of Hg.

Entering pressure.			Pressure at end of rubber tube.			Difference between initial and end pressure.	
Systolic.	Diastolic.	Difference.	Systolic.	Diastolic.	Difference.	Systolic.	Diastolic.
145	50	95	120	60	60	−25	+10
184	86	98	152	104	48	−32	+18
220	125	95	188	148	40	−32	+23

The same general results followed, no matter what the thickness of the wall of the tube experimented on might be, and no matter what its length, but the difference in the case of the thinner walled tubes was even more striking.

Working with a rubber tube 30 cm. long, and with walls 0·2 mm. thick, with an entering pressure of 78 mm. of Hg diastolic and a 148 mm. systolic, an almost continuous pressure of 104 mm. diastolic and 107 mm. systolic was obtained at the end of the resilient tube.

With raised pressure not only was the curve of less amplitude, but its form also was altered, the top becoming flattened and the dicrotic wave less marked, indeed it took on the characters which have frequently been described as occurring in the sphygmograms of cases of high blood pressure.

In order to test the correctness of the supposition that as the general level of pressure was raised the resilience of the wall was increasingly brought into play, a series of experiments was carried out, using the same initial pressures and the same thickness of tube wall, but varying lengths of tube. It was found that lengthening the tube had the same effect of approximating the systolic and the diastolic pressures and making the curve take on the characters of a "high pressure" sphygmogram. For instance, using a tube with walls 0·8 mm. thick, the following results were obtained:—

Length of tube in cm.	Entering pressure.			Pressure at end of rubber tube.			Difference between initial and end pressure.	
	Systolic.	Diastolic.	Difference.	Systolic.	Diastolic.	Difference.	Systolic.	Diastolic.
15	160	40	120	126	60	66	−34	+20
30	160	42	118	118	74	44	−42	+32
60	117	40	117	108	78	33	−49	+38

The same initial pressure differences were then tried on tubes of the same length and calibre, but with walls of different thicknesses, namely, 0·8, 0·6, 0·4, and 0·2 mm., when the same sort of results were obtained, viz., the thinner and consequently the more resilient the tube, the more was the systolic pressure lowered and the diastolic raised by passing through the tube, that is, the nearer the resultant curve approached a straight line.

It was quite remarkable to observe how with an entering pressure such as 160 mm. systolic and 40 mm. diastolic, a curve in the 0·8 mm. tube would have all the characters of a low pressure sphygmogram, great amplitude, sharp rise and fall and very well marked dicrotic wave, while with exactly the same entering pressure the curve in the 0·4 mm., and more so in the 0·2 mm., took on all the characters of a high pressure sphygmogram, slow rise, flat top, slow fall, and slightly marked dicrotism.

L. H. and Martin Flack have since found that the introduction of, say, 6 cm. of cat's carotid artery in place of an equal length of pressure tubing alters the characters of the pulse curve from a low to a high pressure curve. The experiments demonstrating this will be published in full later.

From these experiments conducted by S. R. W. and those of L. H. it seems legitimate to draw these conclusions: the form of curve obtained by a sphygmograph or other instrument recording the pulse is the resultant of two factors, the blood-pressure variations produced by the heart and the resilience of the arterial wall, using the term resilience in the sense defined above.

Much at times has been made of the supposed influence of reflected waves on the pulse curve. It is the resilience of the wall which we believe to be the important factor in modifying the curve, and not the reflection of waves from the periphery.

The blood-pressure measured in any artery by the sphygmometer is likewise the resultant of these two factors, and the measurement does not necessarily give us the full systolic pressure produced by the heart; much of the force is spent in dilating a soft distensile artery. Further, since the character of the flow in an artery largely depends on the resilience of its

walls, it is obvious that, the more resilient or yielding are those supplying any part, the more closely will the blood stream at the threshold of the capillary area supplied approach a uniform pressure (roughly the mean between the systolic and diastolic pressures, less, of course, what has been lost by friction), while the harder or less resilient the arterial wall, the more closely will the variations approach those in the aorta.

Now all the arteries, and to a greater extent the arterioles, are contractile, and under the influence of the nervous system, and with an increased tone or contraction, they become not only narrower as to lumen, but also thicker as to wall, that is less resilient, so it may happen that the organism can with the same heart force vary the pressure at the threshold of a particular capillary area between an intermittent pressure with a high systolic beat and an almost continuous one, with a lower systolic pressure. In the one case there would be a hammer-like percussive wave beating open the capillaries, the blood would be hammered in ; in the other there would be a more continuous pressing in of the blood at a lower tension.

It may be that narrowing of lumen and lessened total flow goes with the more percussive wave due to hardening, but this does not necessarily follow, for it is possible that a tightening of the muscular coat, and a lessening of the resilience, may take place before actual narrowing occurs. It is further possible that the great arteries and the arterioles act differently, or independently in some cases.

We advance the view that the throbbing and capillary pulse observed in an acutely inflamed area is due to an increased tone of the arterial walls, a lessening of their resilience ; this throbbing is often relieved by hot fomentations which act by relaxing the contraction of the vessel walls. In cases of aortic regurgitation the hammer-like pulse propelled through the harder leg arteries secures to the legs an adequate supply of blood, compensating as it does for the diastolic fall due to the regurgitation ; there, again, hot water baths relax the arterial wall.

One of us, L. H., with Martin Flack,* has shown that in the case of the salivary gland each alveolus is surrounded by a tough membrana propria which resists expansion and allows the secreting cells to draw fluid from the capillaries and raise the secretory pressure to almost double the height of the arterial blood-pressure, without obliterating the surrounding capillaries or interrupting the venous outflow. Under such conditions the veins are narrowed, by the expansion of the alveoli up to their limiting membranes, and the blood vessels, arteries, capillaries, and veins form a system of rigid vessels with a rapid rate of flow, the pulse even coming through into the

* 'Roy. Soc. Proc.,' 1912, B, vol. 85, p. 312.

veins, the whole gland feeling tense to the touch. Increased hardness of the arteries, supplying such an active organ, permits the full force of the systolic wave to come into play, and insures a flow of blood in the face of the increased osmotic pressure and swelling of the tissues.

In the condition of local inflammation the heart beats forcibly, and the arteries conduct the full force of the wave to the swollen, tense, inflamed part; the part throbs with pain. A fomentation, by softening the arteries and the confining frameworks which surround the tissue cells, or the surgeon's knife by relieving the tension, permits an ampler flow of blood with its curative properties.

It is the altered osmotic condition of the infected inflamed tissue which causes the swelling, and this may advance to such a degree that the circulation is strangled and the part necrosed; before this happens, however, the full stroke of the heart's systole is conveyed by the hard contracted arteries with hammer-like strokes to the part and forces the blood through the vessels, maintaining the circulation and thus allowing the bacterial poisons to be neutralised up to the utmost possible limit. Probably the hypertrophy of the muscular coat of the arteries in certain pathological conditions is correlated with a need for the hammer-like stroke.